2. Kersten JR, Schmeling TJ, Pagel PS, Gross GJ, Warltier DC. Isoflurane mimics ischemic preconditioning via activation of K(ATP) channels: reduction of myocardial infarct size with an acute memory phase. *Anesthesiology*. 1997;87:361-70.
3. Kehl F, Krolikowski JG, Mraovic B, Pagel PS, Warltier DC, Kersten JR. Is isoflurane-induced preconditioning dose related? *Anesthesiology*. 2002;96:675-80.
4. Piriou V, Chiari P, Lhuillier F, Bastien O, Loufoua J, Raissy O, et al. Pharmacological preconditioning: comparison of desflurane, sevoflurane, isoflurane and halothane in rabbit myocardium. *Br J Anaesth*. 2002; 89:486-91.
5. Toller WG, Kersten JR, Pagel PS, Hettrick DA, Warltier DC. Sevoflurane reduces myocardial infarct size and decreases the time threshold for ischemic preconditioning in dogs. *Anesthesiology*. 1999;91:1437-46.
6. Penta DP, Polisa P, Tomai F, De Paulis R, Turani F, Zupancich E, et al. Recovery of LV contractility in man is enhanced by pre-ischemic administration of enflurane. *Ann Thorac Surg*. 1999;68:112-8.
7. De Hert SG, ten Broecke PW, Mertens E, Van Sommeren EW, De Blier IG, Stockman BA, et al. Sevoflurane but not propofol preserves myocardial function in coronary surgery patients. *Anesthesiology*. 2002;97:42-9.
8. Garcia C, Julier K, Bestmann L, Zollinger A, von Segesser LK, Pasch T, et al. Preconditioning with sevoflurane decreases P-ERK1 expression and improves one-year cardiovascular outcome in coronary artery bypass graft surgery. *Br J Anaesth*. 2005;94: 159-65.
9. Kevin LG, Katz P, Camara AK, Novalija E, Riess ML, Stowe DF. Anesthetic preconditioning: effects on latency to ischemic injury in isolated hearts. *Anesthesiology*. 2003;99: 385-91.
10. Kersten JR, Orth KG, Pagel PS, Mei DA, Gross GJ, Warltier DC. Role of adenosine in isoflurane-induced cardioprotection. *Anesthesiology*. 1997;86:1128-39.
11. Chiari PC, Pagel PS, Tanaka K, Krolikowski JG, Ludwig LM, Trillo RA Jr, et al. Intravenous emulsified halogenated anesthetics produce acute and delayed preconditioning against myocardial infarction in rabbits. *Anesthesiology*. 2004;101:1160-6.

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Reply to the Editor:

My colleagues and I thank Lange and colleagues for pointing out that we omitted mentioning the importance of volatile anesthetics and ischemic preconditioning (IPC) in a number of animal models and possibly human beings. For example, Kersten and coworkers¹ showed that halothane preconditioning in barbiturate-anesthetized dogs led to a profound infarct size reduction ($25.3\% \pm 2.9\%$ to $11.8\% \pm 2.7\%$) and that this reduction

was as effective as IPC ($9.6\% \pm 2.0\%$). We were remiss in not discussing these results and their importance to IPC. Lange and colleagues also stated, "Anesthetic preconditioning significantly reduces arrhythmias after ischemic periods." The antiarrhythmic effect of volatile anesthetics is more problematic. Recently Owczuk and associates² discussed how desflurane can prolong the QTc interval and may result in lethal cardiac arrhythmias. Huneke and coworkers³ also reported that halothane, and to a lesser extent isoflurane and sevoflurane, may lead to ventricular arrhythmias by shortening the refractory period. In addition, volatile anesthetics are known to inhibit a number of voltage-gated potassium ion channels, which could further increase the risk of ventricular tachycardia in predisposed patients.³ It is unfortunate that most animal studies investigating volatile anesthetics and IPC rarely report the incidence and duration of arrhythmias during ischemia-reperfusion. On the other hand, we reported that pretreating the heart with an intravenous solution of the adenosine A1 receptor agonist 2-chloro-N6-cyclopentyladenosine and sodium ion fast-channel modulator lidocaine (AIL) administered 5 minutes before and during 30 minutes of acute regional ischemia in the rat in vivo resulted in no deaths, virtually no severe ventricular arrhythmias and an infarct size equivalent to that seen with IPC.⁴ Finally, Lange and colleagues believe it is unlikely that the administration of a selective A1 agonist plus a volatile anesthetic could further enhance cardioprotection. However, we believe the combination may reduce the amount of each agent needed to exert cardioprotection; that is, the A1 receptor agonist may reduce the effective therapeutic threshold of the volatile anesthetic agent. This concept was recently reported by Chiari and colleagues,⁵ in whose study postconditioning during early reperfusion reduced the concentration of isoflurane necessary to exert cardioprotection. Because the underlying mechanisms of volatile anesthetics and the AIL combination are not fully understood, the synergistic effects of volatile anesthetics and A1 analogs (or AIL) remain to be determined. Moreover, modulating cardiac voltage-gated sodium ion fast channels with local anesthetics has received surprisingly little attention in IPC strategies, despite the resetting of the resting membrane potential toward a

more depolarized state during ischemic injury.³ To quote nineteenth century essayist and politician Benjamin Disraeli, "What we anticipate seldom occurs; what we least expected generally happens."⁵ Importantly, our work has shown that it is the combination of A1 agonist (or adenosine) and lidocaine administration that confers functional, electrical, and metabolic protection during myocardial ischemia-reperfusion.³ It would be of interest to study the effects of volatile anesthetics in the presence of the combination of A1 agonist (or adenosine) and lidocaine during and after acute myocardial ischemia in clinically relevant experimental models.⁶

Geoffrey P. Dobson, MSc, PhD

Department of Physiology and Pharmacology
James Cook University, Townsville,
Queensland, Australia

References

1. Kersten JR, Schmeling TJ, Pagel PS, Gross GJ, Warltier DC. Isoflurane mimics ischemic preconditioning via activation of K(ATP) channels: reduction of myocardial infarct size with an acute memory phase. *Anesthesiology*. 1997;87:361-70.
2. Owczuk R, Wujtewicz MA, Sawicka W, Lasek J, Wujtewicz M. The influence of desflurane on QTc interval. *Anesth Analg*. 2005; 101:419-422.
3. Huneke R, Fassl J, Rossaint R, Luckhoff A. Effects of volatile anesthetics on cardiac ion channels. *Acta Anaesthesiol Scand*. 2004;48: 547-61.
4. Canyon SJ, Dobson GP. Pretreatment with an adenosine A1 receptor agonist and lidocaine: a possible alternative to myocardial ischemic preconditioning. *J Thorac Cardiovasc Surg*. 2005;130:371-7.
5. Chiari PC, Bienengraeber MW, Weihrauch D, Krolikowski JG, Kersten JR, Warltier DC, et al. Role of endothelial nitric oxide synthase as a trigger and mediator of isoflurane-induced delayed preconditioning in rabbit myocardium. *Anesthesiology*. 2005; 103:74-83.
6. Dobson, G P. A chaos of delight: science, religion and myth and the shaping of Western thought. Oakville (CT): David Brown; 2005
doi:10.1016/j.jtcvs.2005.09.042

Management of pediatric airway obstruction

To the Editor:

We read with interest Vinograd and colleagues' article¹ on the management of airway obstruction in children by using self-expandable metal stents (SEMS). We commend the authors on a thorough and detailed description of the difficulties encountered in treating children with this complex problem.